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Ozone, Fine Particulate Matter, and Chronic Lower Respiratory Disease Mortality in the United States

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Abstract

Rationale—Short-term effects of air pollution exposure on respiratory disease mortality are well established. However, few studies have examined the effects of long-term exposure, and among those that have, results are inconsistent.

Objectives—To evaluate long-term association between ambient ozone, fine particulate matter ($PM_{2.5}$, particles with an aerodynamic diameter of 2.5 μm or less), and chronic lower respiratory disease (CLRD) mortality in the contiguous United States.

Methods—We fit Bayesian hierarchical spatial Poisson models, adjusting for five county-level covariates (percentage of adults aged ≥65 years, poverty, lifetime smoking, obesity, and temperature), with random effects at state and county levels to account for spatial heterogeneity and spatial dependence.

Measurements and Main Results—We derived county-level average daily concentration levels for ambient ozone and $PM_{2.5}$ for 2001–2008 from the U.S. Environmental Protection Agency's down-scaled estimates and obtained 2007–2008 CLRD deaths from the National Center for Health Statistics. Exposure to ambient ozone was associated with an increased rate of CLRD deaths, with a rate ratio of 1.05 (95% credible interval, 1.01–1.09) per 5-ppb increase in ozone; the association between ambient $PM_{2.5}$ and CLRD mortality was positive but statistically insignificant (rate ratio, 1.07; 95% credible interval, 0.99–1.14).

Conclusions—This study links air pollution exposure data with CLRD mortality for all 3,109 contiguous U.S. counties. Ambient ozone may be associated with an increased rate of death from CLRD in the contiguous United States. Although we adjusted for selected county-level covariates and unobserved influences through Bayesian hierarchical spatial modeling, the possibility of ecologic bias remains.

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Keywords

air pollution; chronic lower respiratory disease mortality; Bayesian hierarchical spatial models

Short- and long-term exposure to ozone and $PM_{2.5}$ (particles with an aerodynamic diameter of 2.5 μm or less) air pollution may contribute to an increased risk of the onset of disease, exacerbation of symptoms, and mortality (1). The short-term effects of ambient ozone and $PM_{2.5}$ on respiratory disease mortality are well established (2–5). However, few studies have examined the effects of long-term exposure and, among those that have, results are inconsistent (6–11). Understanding the specific contribution of short-term (up to a few weeks) versus long-term (1 yr or more) exposure is complicated and is typically approached using different study designs (1). Unlike time-series studies, which examine deaths due to short-term exposure, cohort studies are used to evaluate deaths over a longer time period, reflecting cumulative effects of both short- and long-term exposure.

Using the American Cancer Society (ACS) cohort, Jerrett and colleagues found a significant association between long-term ozone exposure and respiratory disease mortality (8). In their more recent study of a California component of the ACS cohort, however, the association between ozone exposure and respiratory mortality was positive but insignificant (7). A multicity study of Medicare participants (mainly ≥65 yr) did find a positive association between long-term exposure to ozone and an increased risk of respiratory disease death—particularly in those with chronic obstructive pulmonary disease (COPD), diabetes, congestive heart failure, and myocardial infarction (11). Results from studies evaluating the association between long-term exposure to $PM_{2.5}$ and respiratory disease mortality have suggested no association or positive but insignificant association (9, 10).

Deaths from chronic lower respiratory disease (CLRD), which includes mainly asthma and COPD (emphysema and chronic bronchitis), account for 50% of all respiratory disease mortality and is the third leading cause of death in the United States (12). Although short-term studies suggest a linkage between air pollution and CLRD morbidity and mortality (13), the effects of long-term air pollution on CLRD mortality remain uncertain. Previous studies focus on specific segments of population (e.g., aged ≥65 yr) or individuals willing to participate in prospective cohort studies (e.g., the ACS cohort). However, these studies are limited to metropolitan areas, and no national study exists. The U.S. National Environmental Public Health Tracking Network (Tracking Network) is a nationwide surveillance system that contains environmental and health data at state and county levels (14). In this study, county-level data were used to examine the association between long-term exposure to ozone and $PM_{2.5}$ and CLRD mortality. We restricted our study to 48 states and the District of Columbia (3,109 counties), because modeled estimates of ozone and $PM_{2.5}$ concentration were not available in the noncontiguous states of Alaska and Hawaii. To minimize potential bias related to traditional ecologic analyses, we used Bayesian hierarchical spatial modeling to account for five known place-varying confounders and unobserved heterogeneity and spatial dependence (15).

Methods

We included 265,223 deaths that occurred among adults 45 years of age or older during 2007–2008 in the contiguous United States. Each death record had a U.S. county identifier in the restricted mortality data file, which allowed us to summarize death counts by county and to link them with other county-level data sources. CLRD deaths included those with underlying cause coded as J40–J47 (ICD-10 codes). We derived county-level average daily ozone and $PM_{2.5}$ by aggregating 2001–2008 census-tract-level 8-hour maximum ozone and 24-hour average $PM_{2.5}$ concentration, generated by the U.S. Environmental Protection Agency for the Tracking Network based on monitored data and output from the Community Multi-scale Air Quality modeling system (16). County-level lifetime smoking prevalence (percentage of adults who were current or former smokers) and obesity prevalence (percentage of obese adults with body mass index [the ratio of height to weight] ≥30) were derived from the Behavioral Risk Factor Surveillance System (2007–2008) (17), using the method suggested by Zhang and colleagues (18). The county-level percentage of adults at least 65 years of age and poverty levels (percentage of adults below the federal poverty line) were obtained from 2007–2008 census data (19). Extremely hot days were defined as the average annual number of days with maximum temperature equal to or greater than 90 degrees Fahrenheit (°F). County-level daily maximum temperatures during 2001–2008 were obtained from the Tracking Network, originally from the North American Land Data Assimilation System (14).

We fit Bayesian hierarchical spatial Poisson models using CLRD death counts as the outcome and county-level variables as predictors. Five models were explored with different random effect specifications: model 1, state unstructured random effects only; model 2, state unstructured and county unstructured random effects; model 3, state unstructured and county spatially structured random effects; model 4, state unstructured, county unstructured, and county spatially structured random effects; model 5, model 4 with a mixture parameter embedded between county unstructured and county spatially structured random effects. Epidemiologically, state unstructured random effects specify state-level contextual effects on mortality; county unstructured random effects specify county-level heterogeneous contextual effects whereas county spatially structured random effects capture possible spatial dependence (i.e., spatial autocorrelation between adjacent counties). The mixture parameter allows the balance of county-level heterogeneity and spatial dependence. Our log-link Poisson regression model is $\log[y_i] = \log[E_i] + \alpha + X_i\beta + ST_{j[i]} + \rho_i U_i + (1 - \rho_i)S_i$, where y_i is the number of deaths for county i ($i = 1, \ldots, 3{,}109$), E_i is the population (≥45 yr), α is the intercept, X_i is the vector of seven predictors ($X_{1,i}, \ldots, X_{7,i}$), $\beta_{[1, \ldots, 7]}$ is the corresponding regression coefficient, $ST_{j[i]}$ ($j = 1, \ldots, 49$) is state unstructured random effects, U_i is county unstructured random effects, S_i is county spatially structured random effects, and ρ is the mixture parameter ($0 \le \rho \le 1$). County spatially structured random effects are formulated as $S_i|S_j \sim \text{Normal}(\overline{S}_i, \tau_i^2)(i \neq j)$ (20), where $\overline{S}_i = (1/\sum_j w_{ij})\sum_j S_j w_{ij}, \tau_i^2 = \tau_s^2/\sum_j w_{ij}$, $w_{ij} = 1$, if i, j are adjacent counties, otherwise $w_{ij} = 0$. The state unstructured and county unstructured random effects are formulated as $ST_{j[i]} \sim \text{Normal}(0, \tau_{ST}^2)$ and $U_i \sim \text{Normal}(0, \tau_u^2)$. τ_{ST}^2, τ_s^2, and τ_u^2 are the variance parameters of $ST_{j[i]}$, S_i, and U_i. In full

Bayesian analyses, prior distribution must be specified for these three variance parameters. We assigned diffusive/noninformative gamma distributions for these three parameters, as suggested by Bernardinelli and colleagues (21). We implemented these five models in WinBUGS1.4.3 and used the deviance information criterion (DIC) to compare model fit (15, 22).

Results

Table 1 shows the mean, range, and quartiles of ozone, $PM_{2.5}$, and five selected demographic, socioeconomic, behavioral, and meteorological characteristics. Ozone exposure ranged from 27.8 to 52.0 ppb (median, 41.2 ppb), $PM_{2.5}$ exposure ranged from 4.8 to 16.8 $\mu g/m^3$ (median, 10.9 $\mu g/m^3$), percentage of adults 65 years of age or older ranged from 3.1 to 39.6% (median, 15.1%), percentage of adults below the federal poverty line ranged from 2.7 to 49.5% (median, 12.4%), lifetime smoking prevalence ranged from 24.6 to 68.9% (median, 51.5%), obesity prevalence ranged from 16.6 to 50.2% (median, 30.0%), and extremely hot days ranged from 0 to 197 (median, 46).

Table 2 shows that model 3 produced the lowest DIC. The difference between the DIC for this model (21,474.7) and the DICs for models 4 and 5 (21,475.1 and 21,479.1, respectively) is admittedly small (<5), indicating that any of them could be the best model for describing the data (22). Still, model 3, with state unstructured and county spatially structured random effects, is preferred because it contains fewer parameters (23). In contrast, the difference is substantial between the DICs for models 3, 4, and 5 (21,474.7, 21,475.1, and 21,479.1, respectively) and the DICs for models 1 and 2 (21,606.4 and 21,607.4, respectively) (>5). It is evident that county spatially structured random effects dominate spatial dependence between neighboring counties, reflecting the effects of unobserved, spatially structured covariates.

Bayesian inference is based on posterior means (analogous to means) and credible intervals (CIs, analogous to confidence intervals). Table 3 presents adjusted rate ratios (RRs) and 95% CIs from model 3 (the preferred model with state unstructured and county spatially structured random effects), measured per five-unit increment for all variables. All predictors were positively associated with CLRD deaths. Specifically, the RR was 1.05 (95% CI, 1.01–1.09) per 5-ppb increase in ozone exposure.

Ozone and $PM_{2.5}$ were associated with a 5% (per 5-ppb increase in average ozone) and a 7% (per 5-$\mu g/m^3$ increase in average $PM_{2.5}$) increase in CLRD mortality, respectively, although the association between $PM_{2.5}$ and CLRD mortality was not statistically significant. Together, ozone and $PM_{2.5}$ explained about 3% of the total variation in log RRs (Table 4). Table 4 also shows that all predictors combined explained about 35% of the total variation with lifetime smoking, age (adults aged ≥65 yr), and poverty explaining most, whereas other unobserved covariates at state (5%) and county (60%) levels explained about 65%.

Discussion

Our principal finding is that after controlling for selected demographic, socioeconomic, behavioral, and environmental risk factors, and other spatially unstructured and structured

contextual influences, ozone is associated with increased CLRD mortality rates across U.S. counties. A few cohort studies have observed similar results for ozone, but they were limited in terms of demographic or geographical coverage (8, 11, 24). This nationwide study explored the linkage between county-level concentration levels and aggregated deaths across the contiguous United States. Our analyses differ fundamentally from traditional ecologic analyses in that we used Bayesian hierarchical spatial modeling. Bayesian modeling allows for direct control of known (i.e., percent adults aged ≥65 yr, lifetime smoking, poverty, obesity, and temperature) and unknown (unstructured and spatially structured) risk factors. These analyses showed that CLRD mortality was significantly associated with ozone exposure. Such correlation might reflect an amalgam of complex pathophysiological pathways through which ozone could induce or accelerate pulmonary inflammation leading to CLRD mortality (25, 26).

Our results for the long-term effects of ozone on CLRD mortality are generally consistent with the findings from the ACS cohort (448,850 participants in 86 U.S. metropolitan areas during 1977–2000) (8) and Medicare subpopulations (3,210,511 persons with COPD in 105 U.S. cities during 1985–2006) (11). Our adjusted RR estimate of 1.05 per 5-ppb increase in ozone—which is equivalent to 1.10 per 10-ppb increment— is lower than the estimate from Medicare participants hospitalized with COPD (RR, 1.07 [95% CI, 1.05–1.10] per 5-ppb increment), but it is higher than that from the ACS cohort (RR, 1.04 [95% CI, 1.01–1.07] per 10-ppb increment). The study of a California ACS cohort reported a positive albeit insignificant association (RR, 1.02 [95% CI, 0.90–1.15] per 10-ppb increment), which might be due to the small number of participants (n = 73,711). The difference in RR estimates could be due in part to the difference in participants or study areas included in these studies. Participants hospitalized with COPD in the Medicare study might have had higher risk of CLRD death due to ozone exposure than did people without preexisting COPD as well as the general U.S. population. Participants in the ACS study were mainly white with relatively high educational attainment (27). Thus, subjects in the ACS study might have been healthier, and have had lower risk of CLRD death due to ozone exposure, than the U.S. population generally.

We found a positive but statistically insignificant association between long-term $PM_{2.5}$ exposure and CLRD mortality, with an adjusted RR estimate of 1.07 (95% CI, 0.99–1.14) per 5-$\mu g/m^3$ increase in $PM_{2.5}$ exposure, which is equivalent to 1.14 per 10-$\mu g/m^3$ increment. The Harvard Six Cities cohort (8,096 white participants) and California ACS cohort (73,711 participants) resulted in similar findings, with adjusted RRs of 1.05 (95% CI, 0.95–1.15) for the California cohort and 1.08 (95% CI, 0.79–1.49) for the Harvard Six Cities cohort for an increase of 10 $\mu g/m^3$ in $PM_{2.5}$ exposure (7, 9). A study of a large ACS cohort (448,850 participants) using a single-pollutant model reported a similar association (RR, 1.03 [95% CI, 0.96–1.11]) but reported an inverse and insignificant association in a two-pollutant model when ozone was included (RR, 0.93 [95% CI, 0.84–1.03]) (8). Further studies are needed to confirm the association between $PM_{2.5}$ and CLRD mortality at both the individual and aggregated population levels.

The increased risk of death due to CLRD associated with ozone was small compared with the risk posed by lifetime smoking, older age (≥65 yr), and temperature. Nevertheless, the

positive association between air pollution and CLRD mortality persisted after controlling for known and unknown factors at county and state levels. The adjusted RR for ozone was close to that for poverty, a county-level socioeconomic indicator—although lifetime smoking and older age were two known leading contributors to the CLRD mortality variation. It is worth noting that county-level unknown, spatially correlated influences contributed more than half of the CLRD death variations across U.S. counties; these influences were not considered in most previous studies. Also, the inclusion of spatially correlated random components could potentially increase the precision of the RR estimate for the known risk factors in which we were interested.

This study has several limitations. First, our single ecological study could not make any causal inference. Although we adjusted for available county-level covariates and unobserved influences, the possibility of ecologic bias remains. Furthermore, including 8 years of exposure data could be one of the strengths; however, it could also be a limitation because people could move during this time period and disease latency could be longer than the years we included in this study. Similarly, we could not account for any seasonal variation in or trend of exposure during this time period. National annual average ozone and $PM_{2.5}$ both showed downward trends from 1980 (for ozone) and 2000 (for $PM_{2.5}$), but such trends are not smooth and do show year-to-year influences of weather conditions, which contribute to ozone and $PM_{2.5}$ formation in the air (28, 29). We used 8-year (2001–2008) average ozone and $PM_{2.5}$ as proxies for their long-term exposure estimates; however, using exposure averaged during the early years (2001–2002) did not meaningfully change county-level RR estimates (*see* Table E4 in the online supplement). In addition, we did not address the seasonality of CLRD deaths, ozone, and $PM_{2.5}$ because of the small sample size of CLRD deaths by season at the county level. Ozone shows a clear seasonal pattern, and linking the seasonal timing of death might strengthen the association found, if the number of deaths by season at the county level were sufficient to allow us to do so. Finally, we could not evaluate the sensitivity of ICD-10 codes (J40–J47) for the diagnosis of CLRD. Potential misclassification of CLRD as an underlying cause of death might introduce additional uncertainties in our findings. Findings of this national study suggest that ozone and $PM_{2.5}$ might have contributed to increased CLRD mortality across U.S. counties, although residual confounding cannot be excluded. The association observed between long-term ozone exposure and CLRD mortality across U.S. counties is in line with findings from previous cohort studies, but this study expands the evidence to the U.S. population. The positive association between long-term $PM_{2.5}$ exposure and CLRD mortality is consistent with findings from previous studies. This U.S. national study provides additional evidence that ambient air pollutants, particularly ozone, could be important contributing factors in CLRD mortality.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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At a Glance Commentary

Scientific Knowledge on the Subject

The short-term effects of ambient ozone and fine particulate matter ($PM_{2.5}$, particles with an aerodynamic diameter of 2.5 μm or less) on respiratory disease mortality are well established. However, few studies have examined the effects of long-term exposure and, among those that have, results are inconsistent.

What This Study Adds to the Field

This nationwide study links air pollution exposure data of ambient ozone and $PM_{2.5}$ with chronic lower respiratory disease mortality for 3,109 contiguous U.S. counties. Our findings suggest that long-term exposure to ambient ozone may be associated with an increased rate of death from chronic lower respiratory disease in the contiguous United States.

Table 1

Distribution of County-Level Ozone, $PM_{2.5}$, and Demographic, Socioeconomic, and Behavioral Characteristics among 3,109 Contiguous U.S. Counties

Variable	Mean	Range	Min	Q1	Median	Q3	Max
Adults aged ≥65 yr, %	15.5	36.5	3.1	12.8	15.1	17.8	39.6
Poverty, %	13.3	46.8	2.7	9.6	12.4	15.9	49.5
Lifetime smoking, %	51.5	44.2	24.6	49.1	51.5	54.0	68.9
Obesity, %	30.3	33.6	16.6	28.1	30.0	32.4	50.2
Extremely hot days (≥90°F)	53.8	197.0	0.0	20.3	46.3	78.6	197.0
Ozone, ppb	40.9	24.2	27.8	39.0	41.2	42.9	52.0
$PM_{2.5}$, μg/m^3	10.7	12.0	4.8	8.7	10.9	12.5	16.8

Definition of abbreviations: Max = maximum; Min = minimum; $PM_{2.5}$ = particulate matter with an aerodynamic diameter of 2.5 μm or less; Q1 = first quartile; Q3 = third quartile.

Ozone and $PM_{2.5}$ exposure were averaged during 2001–2008.

Table 2

Comparison of Deviance Information Criterion for Models with Different Random Effect Specification

Model	Random Effect Specification	DIC
1	State unstructured random effects only	21,606.4
2	State unstructured and county unstructured random effects	21,607.4
3	State unstructured and county spatially structured random effects	21,474.7
4	State unstructured, county unstructured, and county spatially structured random effects	21,475.1
5	Model 4 with a mixture parameter	21,479.1

Definition of abbreviation: DIC = deviance information criterion.

Table 3

Adjusted Rate Ratios of Predictors Associated with Chronic Lower Respiratory Disease Deaths Measured per Five-Unit Increment for All Variables

Variable	Rate Ratio	95% CI
Adults aged ≥65 yr, %	1.09	1.07–1.11
Poverty, %	1.06	1.04–1.08
Lifetime smoking, %	1.13	1.10–1.15
Obesity, %	1.03	1.01–1.05
Extremely hot days (≥90°F)	1.01	1.00–1.01
Ozone, ppb	1.05	1.01–1.09
$PM_{2.5}$, μg/m^3	1.07	0.99–1.14

Definition of abbreviations: CI = credible interval; $PM_{2.5}$ = particulate matter with an aerodynamic diameter of 2.5 μm or less.

Table 4

Variation in Chronic Lower Respiratory Disease Death Rates as Explained by Predictors and Random Effects

Variable	Mean	2.50%	Median	97.50%
Adults aged ≥65 yr, %	7.8%	4.9%	7.7%	11.1%
Poverty, %	5.7%	3.1%	5.6%	8.8%
Lifetime smoking, %	14.6%	10.0%	14.5%	19.7%
Obesity, %	0.8%	0.0%	0.7%	2.3%
Extremely hot days (≥90°F)	3.0%	0.2%	2.6%	7.8%
Ozone, ppb	1.3%	0.0%	1.1%	3.6%
$PM_{2.5}$, $\mu g/m^3$	1.8%	0.0%	1.5%	5.8%
State random effects	4.9%	2.5%	4.7%	8.5%
County spatial random effects	60.1%	53.7%	60.2%	66.0%

Definition of abbreviation: $PM_{2.5}$ = particulate matter with an aerodynamic diameter of 2.5 μm or less.